Exhibit D

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BEFORE THE POLLUTION CONTROL HEARINGS BOARD
IN AND FOR THE STATE OF WASHINGTON

DIOXIN/ORGANOCHLORINE CENTER, COLUMBIA RIVER UNITED, INC., and PUGET SOUND ALLIANCE, Appellants, v. WASHINGTON STATE DEPARTMENT OF ECOLOGY, CHRISTINE O. GREGOIRE, in her official capacity as Director of Washington State Department of Ecology, and the STATE OF WASHINGTON, Respondents.	No. 91-186 and
JAMES RIVER II, INC., et al., Appellants, v. STATE OF WASHINGTON, DEPARTMENT OF ECOLOGY, Respondents, and DIOXIN/ORGANOCHLORINE CENTER, COLUMBIA RIVER UNITED, INC., and PUGET SOUND ALLIANCE, Intervenors-Respondents.	PCHB No. 91-140 PCHB No. 91-143 PCHB No. 91-146 PCHB No. 91-147 PCHB No. 91-148 PCHB No. 91-150 PCHB No. 91-151 PCHB No. 91-154 PCHB No. 91-169 PCHB No. 91-182 DECLARATION OF DONALD C. MALINS IN SUPPORT OF APPELLANTS/ INTERVENORS' MOTION FOR SUMMARY JUDGMENT

I, DONALD C. MALINS, declare as follows:

1. My name is Donald C. Malins. I hold Doctor of Philosophy and Doctor of Science degrees in biochemistry. I am currently Head of the Environmental Biochemistry Program at the Pacific Northwest Research Foundation, an independent, non-profit medical research facility, located in Seattle, Washington. I am an expert in the fields of biochemistry and toxicology, and my professional work has focused particularly on the effects of environmental chemicals on aquatic organisms and the etiology of cancer.

2. Other posts which I presently hold are an affiliate professorship in the University of Washington's Department of Environmental Health and a Research Professorship in Chemistry at Seattle University. I am one of the founders of, and have been for many years the Editor-in-Chief of, the international journal Aquatic Toxicology. I have also served as a U.S. member of the Science Advisory Board of the International Joint Commission for the Great Lakes. A copy of my curriculum vitae is attached hereto as Exhibit A and is incorporated herein by reference.

3. Since 1967, my principal professional work has involved field and laboratory studies of the effects of environmental chemicals on aquatic organisms. In the last four years I have also been concerned with the role played by environmental chemicals in the causation of cancer in humans. I have specialized knowledge of the toxicology of chlorinated hydrocarbons, including the chemicals known as polychlorinated dibenzo-p-dioxins (including 2,3,7,8 tetrachlorodibenzo-p-dioxin,

or 2,3,7,8-TCDD) and other chlorinated hydrocarbons which are measured as adsorbable organic halides (AOX) that are waste-products of the chlorine bleaching process in the pulp and paper manufacturing industry. In the course of my professional work, I have scientifically investigated the effects of chlorinated hydrocarbons in the environment and reviewed many scientific studies of the toxicological effects of chlorinated hydrocarbons as well.

4. It is my professional opinion that the discharges to the aquatic environment of chlorine bleaching pulp and paper mill wastes, particularly 2,3,7,8-TCDD and AOX, will likely significantly harm the environment and a broad spectrum of organisms that rely on it. These organochlorine compounds are causally linked to a wide variety of adverse effects in the environment, organisms dependent on that environment, and human beings. Furthermore, discharges of 2,3,7,8-TCDD and AOX occur to receiving and adjacent ecosystems which likely are already significantly contaminated with toxic substances. Thus, the substantial toxic discharges from the chlorine bleaching pulp and paper industry in Washington State most probably have caused and likely will continue to cause significant adverse environmental damage, and have also increased the cumulative toxic threat to the environment.

TOXICITY OF 2,3,7,8-TCDD

5. Chlorine bleaching pulp and paper mills discharge polychlorinated dibenzo-p-dioxins (PCDDs or dioxins) and polychlorinated dibenzo-p-furans (PCDFs or furans), among many

other toxic organochlorine compounds. AOX is currently the accepted scientific measure of the total adsorbable organic halides present in chlorine bleaching pulp and paper mill effluent that is preferred among scientists and regulators throughout the world [25]. AOX includes approximately 300 to 1,000 separate organochlorine compounds [18].

6. Of the PCDDs, 2,3,7,8-TCDD is likely the most toxic. 2,3,7,8-TCDD is among the most potent animal carcinogens and is one of the most potent reproductive toxins known [16]. 2,3,7,8-TCDD has been causally linked to adverse effects in human beings, which include reproductive and developmental impairment [2, 3], cytogenetic changes [4, 5], cancer [5, 22, 31, 36, 37, 40], immune system dysfunction [6, 22], neurologic damage and neuropsychological effects [38, 39], dermatologic abnormalities [20], cardiovascular disorders [40], and altered lipid metabolism [40].

7. Human epidemiologic and related human studies have revealed that 2,3,7,8-TCDD is causally related to cancers in a variety of tissues; that is, it has a general carcinogenic effect. Specifically, exposure to 2,3,7,8-TCDD has been causally related to soft tissue sarcomas, non-Hodgkin's lymphoma, Hodgkin's disease, leukemias, lymphomas, biliary cancer, and cancers of the brain, stomach, liver, colon, rectum, prostate, pancreas, kidney, and respiratory system [5, 22, 31, 36, 37, 40].

8. The 2,3,7,8-TCDD has toxic effects on other life forms (e.g. primates, rodents) as well. These include carcinogenesis

[20], teratogenesis [22], immune systems effects [6, 22], reproductive and developmental damage [3, 21, 22], liver damage and hyperplasia [22], hormonal and metabolic effects [22], wasting syndrome [7], neurological impairment and behavioural effects [17, 21], chloracne [22], enzyme induction [22], and altered gene transcription [30].

9. For some sensitive life stages a no observable adverse effects level (NOAEL) for 2,3,7,8-TCDD has not been established. For example, a NOAEL for the chicken embryo, which is the most sensitive life stage for this avian species, has not been attained [15]. Thus, by analogy, the concentration that results in essentially a lack of toxic effect in aquatically dependent birds is uncertain, but nonetheless may occur at the very low concentrations that Ecology allows to be discharged under the wastewater discharge permits it issued to pulp and paper mills.

10. The 2,3,7,8-TCDD is one of a family of related dioxin compounds which produces toxic responses mediated by an intracellular protein, called the Ah receptor [16]. When dioxin or one of the dioxin-like substances attaches to the Ah receptor in an organisms's cell, it causes particular regulatory and structural genes to be transcribed in the nucleus of the cell, inducing the production of several drug-metabolizing enzymes [22]. One of the enzymes induced in large quantities is aryl hydrocarbon hydroxylase (AHH), which can serve as an indicator of an organism's exposure to 2,3,7,8-TCDD [16].

11. It is generally accepted that chemicals that bind to the Ah receptor will produce the same spectrum of toxic effects

as 2,3,7,8-TCDD [20]. This concept has led to the use of "toxic equivalencies" or "TEQs" by the Food and Drug Administration, EPA, Ecology, and other regulatory agencies. The equivalency approach uses the relative toxicities of compounds that bind to the Ah receptor to provide an exposure value expressed in terms of 2,3,7,8-TCDD toxicity [20].

12. The 2,3,7,8-TCDD has a toxicological effect in both humans and other organisms at extremely low doses. There is no scientific evidence that there is a "safe" level of 2,3,7,8-TCDD [20]. That is, the presence of even trace amounts of 2,3,7,8-TCDD in an organism will likely produce some adverse toxicological effect.

TOXICITY OF OTHER ORGANOCHLORINES IN CHLORINE BLEACHING PULP AND PAPER MILL WASTE

13. Furthermore, 2,3,7,8-TCDD is only one of many hundreds of toxicologically significant compounds created as waste products of the chlorine bleaching process of pulp and paper production. These include chloroform, chlorinated guaiacols, resin acids, carbon tetrachloride, and a plethora of other potentially toxic substances [23, 27].

14. A wide variety of studies have been conducted with aquatic organisms exposed to chlorine bleaching pulp and paper mill organochlorine wastes. These studies reveal the significant adverse effects of these compounds on aquatic organisms. Among the responses to exposure to pulp and paper mill wastes are alterations in growth and reproduction; embryo and fry mortalities; vertebral and spinal deformities; dysfunctions in steroid synthesis; liver damage; and fin rot and apical gill

swelling in fish [25]. Egg numbers and larval growth of the fish the bleny (Zoarces vivaparous) are adversely affected by extremely low concentrations (0.5%) of untreated bleached mill effluent [24]. Trout exposed as far away a 6-11 kilometers from a bleached pulp mill outlet showed related adverse effects to blood hemoglobin and plasma protein concentrations [26]. Thus, the discharge of 2,3,7,8-TCDD or AOX to the surface waters of Washington state from chlorine bleaching pulp and paper mills will likely cause adverse effects to aquatic organisms, even at low concentrations or from distant sources.

15. Other toxicological effects of organochlorines in chlorine bleaching pulp and paper mill effluent on organisms include lethality, reduced survivorship, reproductive effects, delayed sexual maturation, developmental defects, metabolic effects, lesions of the skin and internal organs, immune system disorders, and enzyme induction [18, 19, 22, 25, 26].

16. It is well known that a number of these organochlorine compounds act interdependently, as do other organic compounds [9]. That is, there are additive, synergistic, and antagonistic effects potentially associated with the complex chemical mixtures present in pulp and paper mill effluent and receiving waters. As an example, significant induction of hepatic 7-ethoxyresorufin deethylase (EROD) and AHH occurred in mice as a result of the synergistic interaction of 2,2,4,4',5,5'-hexachlororbiphenyl (HCBP) with 2,3,7,8-TCDD [9]. A failure to account for 2,3,7,8-TCDD and other contaminants that already exist in aquatic ecosystems and organisms therefore underestimates the risks to

these ecosystems and organisms, as well as to those that consume these organisms.

PERSISTENCE, BIOACCUMULATION, AND BIOMAGNIFICATION OF ORGANOCHLORINES

17. The 2,3,7,8-TCDD and other organochlorines are also persistent within the environment and living tissues. Some studies have documented that the toxicokinetic half-life in human tissue is approximately 5 to 8 years [22]. The persistence of 2,3,7,8-TCDD and other organochlorines in soil and sediments is estimated to be longer than this. One study revealed that workers exposed during the 1950's and 1960's had measurably high concentrations of 2,3,7,8-TCDD in their fat when measured in the 1980's [22].

18. Furthermore, 2,3,7,8-TCDD and other organochlorines bioaccumulate in living tissues and biomagnify up the food chain. By these respective mechanisms, older organisms may carry a significant body burden of these compounds, and predators further up the food chain may ingest and retain greater quantities of these compounds. In addition, bioaccumulation factors vary widely among mammals, fishes, birds, and invertebrates and thus some species may accumulate appreciably more 2,3,7,8-TCDD in their tissues than others.

EXISTING CONTAMINATION OF RECEIVING ECOSYSTEMS

19. The fresh and marine waters of Washington state to which chlorine bleaching pulp and paper mills discharge toxic organochlorine compounds include the Columbia, Chehalis, and Snohomish Rivers; Everett, Gray's, and Port Angeles Harbors; Commencement, Bellingham, and Port Gardner Bays; and the Strait

of Juan de Fuca [35]. These water bodies have been and continue to be subjected to tremendous environmental stress. Myriad chemical contaminants have been discharged into these waters over time, and in some areas these contaminants are occurring at levels that are causing chronic toxic effects to aquatic species there [10, 32]. Any additional input of the toxic compounds in chlorine bleaching pulp and paper mill effluent, particularly 2,3,7,8-TCDD and AOX, will likely cause significant adverse effects to the receiving and adjacent ecosystems and to a wide variety of aquatically dependent organisms.

20. In September 1991, EPA corroborated the extent of existing organochlorine contamination within Puget Sound. EPA investigated the extent of PCDD and PCDF contamination in three species of crab, and determined the human cancer risk from consumption of that contaminated crab. EPA concluded that consumption by a 70 kg individual of approximately one third of a pound of Puget Sound crab muscle tissue per week for 30 years results in a cancer risk of up to 5 in 100,000 from that exposure alone [32, hereafter Crab Study]. Consumption of one pound of Puget Sound crab per week yields a cancer risk of up to 1 in 10,000, while the cancer risk that EPA considers "acceptable" is 1 in 1,000,000. Moreover, even the reference samples from a supposedly uncontaminated area of Puget Sound showed significant PCDD and PCDF contamination, and the corresponding cancer risk from consumption of crab from this area was nearly as high as that from the known contaminated areas [32]. As the Crab Study reveals, Ecology's permitted 2,3,7,8-TCDD and AOX discharges are

not being added to essentially pristine environments. The fact is, Puget Sound and many of the organisms present there are already significantly contaminated with PCDDs, PCDFs, and a wide variety of other toxic chemicals [12]. Moreover, consumption of Puget Sound crab and other fishery resources from contaminated areas may significantly increase human cancer risk.

21. Four of the eleven chlorine bleaching mills in Washington State discharge into the Columbia River [35]. With regard to the Columbia River, Ecology and EPA have already determined that fishes there are significantly contaminated with dioxins and furans [1, 33, 34]. The Washington State Department of Health has issued a health advisory for consumption of dioxin and organochlorine contaminated fishes in portions of the upper Columbia River [29].

22. In March 1991, Ecology detected the family of toxic polychlorinated organics discharged from chlorine bleaching pulp and paper mills known as tetrachlorodibenzo-p-furans (TCDFs) in all species of fish tested in the Lake Roosevelt area of the Columbia River [33]. Ecology noted that "[t]he level of TCDF in Lake Roosevelt fish, especially lake whitefish and sturgeon, is very high from both a local and national perspective" [33]. A toxic equivalencies approach uses the relative toxicities of compounds that bind to the Ah receptor, as does 2,3,7,8-TCDD, to provide an exposure value expressed in terms of 2,3,7,8-TCDD toxicity [20]. The TEQs for lake whitefish and sturgeon from Lake Roosevelt "rank among the top 10% of TEQs in EPA's national fish survey....[and] [t]he biological significance of these

findings for fish and their predators in Lake Roosevelt is not known" [33]. Therefore, there is substantial toxic organochlorine contamination already in the Columbia River Basin, and the continued discharge of the toxic compounds in pulp and paper mill effluent will cause further significant environmental damage.

23. In a more recent scientific survey in November 1991, Ecology noted that "[w]hen compared to background, mean TEQ concentrations in resident fish samples from the Columbia mainstem are moderately to substantially elevated" [34, hereafter Sportfish Study]. "Mean TEQ concentrations in channel catfish, carp, and white sturgeon range from 45 to 93 times higher than background" [34]. In conclusion, the Sportfish Study recommended that Ecology "[c]onduct a study to determine the ecological implications of PCDDs/PCDFs in the Columbia River. Investigations to date have only considered potential impacts to human health" [34]. To my knowledge, such a study investigating the ecological implications of this contamination has not been undertaken.

24. With regard to other toxic substances in the Columbia River Basin, elevated levels of DDE, PCBs, and mercury have been found in the eggs of bald eagles, a threatened species in Washington and Oregon [10, 11]. Accumulations of PCBs and DDE have also been reported in mink, river otters, and harbor seals from the lower Columbia River [11]. Indeed, the PCB residues found in mink exceeded concentrations shown to be associated with reproductive impairment in these mammals [11].

25. Toxicologically, chemically stressed systems such as the fresh and marine waters into which the mills discharge are less resilient in the face of an added environmental insult such as that imposed by the addition of toxic organochlorines in pulp mill effluent. An organism exhibiting no toxicological response under laboratory conditions to a given concentration of contaminant, may, under these stressed conditions, be highly sensitive to the same concentration in a toxic milieu. Studies which I, as well as other scientists, have conducted establish clear correlations between the accumulation of toxic chemicals and serious biological effects in aquatic organisms [12]. Just as rats fed fish contaminated with PCBs, dioxin, and other organochlorines exhibit increased reactivity to adverse events (e.g. negative behavioural changes), compared to rats fed uncontaminated diets [17], it is likely that aquatic organisms will be affected by the chemical stresses occurring through their diets, particularly when the affected organisms already exhibit indications of toxic stress from accumulated chemicals contamination.

26. The accumulation of 2,3,7,8-TCDD, other organochlorines, DDE, PCBs, and other contaminants already present in the environment and aquatic organisms clearly suggests that there is no reliable or established margin of safety on which Ecology could rely in issuing permits that allow the continued discharge of 2,3,7,8-TCDD and AOX from chlorine bleaching pulp and paper mills. This is especially true in light of well-established effects on toxicity from interactions among

different types of toxic compounds.

27. The aquatic ecosystems into which pulp and paper mills discharge are complex biological systems which cannot be compared to the limited controlled conditions in a laboratory experiment. Thus, any analysis of the effects of these discharges must take into account the additive, synergistic, and antagonistic effects of 2,3,7,8-TCDD and AOX present in the wastewater discharged, as well as those of the existing contamination in receiving and adjacent waters.

28. Even if it could be shown that dioxin would behave in the real world environment of highly complex chemical interactions as it does in isolation in the laboratory, there is no reason to believe that the 0.013 parts per quadrillion standard would be adequate to protect aquatic organisms and wildlife--it is, in fact, little more than an assumption. In fact, any additional discharge of 2,3,7,8-TCDD or AOX to the surface waters of Washington state will likely contribute to environmental damage and exacerbate the harm from contamination that is already present.

29. There are often vast differences in the ability of organisms to bioconcentrate toxic organic compounds, as well as in their physiological responses to a given contaminant. Thus, it may be that certain species, particularly in their sensitive, early developmental stages, will be affected at the exposure concentrations allowed by the wastewater discharge permits. Moreover, given the additive and synergistic effects of other toxic compounds, and the paucity of information on relevant

effects thresholds, it is highly questionable whether there is a credible scientific basis to permit the discharge of any 2,3,7,8-TCDD or AOX to Washington State waters.

HUMAN CANCER RISKS

30. Ecology has not adequately investigated, analyzed, or addressed thoroughly either the environmental damage or the human cancer risks from its permitting of discharges of 2,3,7,8-TCDD and AOX from pulp and paper mills in this state. Certain segments of the population, notably Native Americans, Asian-Americans, and low-income individuals who subsistence fish, may consume substantially more fish than has been previously estimated, and thus suffer a significantly higher cancer risk [28]. To my knowledge, Ecology has not investigated the human health risks to either the general human population or to these especially sensitive subpopulations from exposure to organochlorines.

31. Furthermore, the susceptibility to cancer varies among human age groups, with children being especially at risk. Yet, Ecology has not investigated how its regulation of pulp and paper mill discharges will impact in any way human populations, including these sensitive populations.

CONCLUSION

32. The continued discharge of 2,3,7,8-TCDD and AOX into the surface waters of Washington State from chlorine bleaching pulp and paper mills will likely significantly adversely affect the health of the receiving waters, and potentially a wide spectrum of the aquatic organisms normally present there, as well

as other dependent life forms. Ecology failed to adequately investigate, analyze, and address the biological consequences of permitting the discharge of 2,3,7,8-TCDD and AOX. Likewise, Ecology failed to account for the toxicological stresses imposed by existing contamination and failed to address the risks posed to the human population by the consumption of contaminated organisms and other exposure to organochlorines.

I declare under penalty of perjury that the foregoing is true and correct to the best of my knowledge. Executed this 31st day of March, 1992, in Seattle, Washington.

Donald C. Malins

DONALD C. MALINS

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EDUCATION

1976	University of Aberdeen, Scotland, DSc
1967	University of Aberdeen, PhD (Biochemistry)
1956	Seattle University, BS (Chemistry)
1953	University of Washington, BA (Anthropology)

PROFESSIONAL POSITIONS

1992-present	**Head, Molecular Epidemiology Program, Pacific Northwest Research Foundation**
1990-1992	Scientific Consultant, National Oceanic and Atmospheric Administration (NOAA) - Natural Resource Damage Assessment (NRDA) legal cases.
1990-1991	United States Member, Great Lakes Science Advisory Board, International Joint Commission.
1989-1991	Senior Scientific Consultant, US Department of Justice, Washington, DC: Exxon Valdez Legal Case.
1989-1991	Member, Board of Directors: American Oceans Campaign
1987-1992	Head, Environmental Biochemistry Program, Pacific Northwest Research Foundation, Seattle, WA
1984-present	Affiliate Professor, Department of Environmental Health, School of Public Health and Community Medicine, University of Washington, Seattle, WA
1980-present	Editor-in-Chief, *Aquatic Toxicology* (Elsevier Biomedical Press, Amsterdam)
1974-1991	Affiliate Professor, School of Fisheries, College of Ocean and Fishery Sciences, University of Washington, Seattle, WA
1974-1987	Director, Environmental Conservation Division, National Marine Fisheries Service, National Oceanic and Atmospheric Administration (NOAA), Seattle, WA
1972-present	Research Professor, Department of Chemistry, Seattle University, Seattle, WA

HONORS AND AWARDS

Examples

United States Department of Commerce Gold Medal, 1982 (for distinguished contributions to marine science)

Doctor of Science (DSc), University of Aberdeen, 1976 (for 20 years of "high distinction" and accomplishment in studies of the chemistry and biochemistry of aquatic systems)

Arthur S. Fleming nomination (Washington, DC), 1969 (recognition as one of "top twenty" scientists under forty in U.S. Government; sponsored by U.S. Civil Service Commission and Washington, DC Jaycees)

PROFESSIONAL ORGANIZATIONS

American Society for Biochemistry and Molecular Biology
American Chemical Society

PROFESSIONAL INTERESTS

Toxicology and biochemistry: (1) Studies of the uptake, disposition and metabolism of toxic chemicals in relation to induced injury at the molecular level (e.g., DNA), and (2) The development of chromatographic and spectrometric analyses of DNA structures in relation to cancer etiology, cancer prediction and risk assessment. Present emphasis includes the etiology of breast cancer and the development of methods for identifying changes in the DNA base structure of the breast.

PROFESSIONAL ACTIVITIES

Miscellaneous

- Principal Investigator, DNA biomarkers in ecological impact assessments, Superfund Program, University of Washington, Seattle (Dr. David Eaton, Program Manager and P.I.), 1992-1995.
- Principal Investigator, DNA lesions in Medaka (O. *latipes*): Development of a micro-method for tissue analysis using gas-chromatography/mass spectrometry, U.S. Army Research and Development Laboratory, Fort Detrick, MD, 1991-1995.
- Principal Investigator, Assessment of modifications in Medaka (O. *latipes*) exposed to chemicals in contaminated groundwater; U.S. Army Research and Development Laboratory, Fort Detrick MD, 1988-1991.
- Editor (with G. Ostrander), Cellular and molecular changes in aquatic systems exposed to toxic chemicals. Lewis Publishers (in press, 1993).
- Member, Review Board, School of Environmental Sciences, University of Sterling, Scotland, November, 1990
- Tour Speaker, American Chemical Society, 1984-1985; 1986
- Co-Editor with JR Sargent, Volume I-IV, "Biochemical and Biophysical Perspectives in Marine Biology," (Academic Press, London), 1974-1978
- Editor, Effects of Petroleum on Arctic and Subarctic Marine Environments and Organisms, Volumes I and II. Vol. I. Nature and Fate of Petroleum; Vol. II. Biological Effects, (Academic Press, New York, 1977)

Chairman, Toxic Chemicals and Aquatic Life: Research and Management, International Conference, Westin Hotel, Seattle WA, September 16-18, 1986.

Presentations, Seminars, and Lectures

Recent examples

Lecture, "The identification of DNA biomarkers in relation to carcinogenesis", U.S. Army Research and Development Laboratory, Fort Detrick, Md., April 1992.
Seminar, "The identification of DNA biomarkers in relation to breast cancer", The Laboratory of Pathology of Seattle, WA, January 1992.
Lecture, "DNA modifications in relation to cancer," Laboratory of Comparative Carcinogenesis, National Cancer Institute, Frederick, MD, April 1991.
Departmental lecture, "Etiology of Cancer in Teleosts," Zoology Department, Oklahoma State University, Stillwater, OK, January, 1991.
Plenary Lecture, "Toxic effects of crude oil on aquatic life," Society of Toxicology, Chicago, May, 1990.
Departmental Seminar, "Alterations in DNA from exposure to environmental chemicals," Environmental Toxicology Department, University of Maryland, Baltimore, May, 1989.
Departmental Lecture, "DNA modifications from exposure to environmental chemicals," Department of Environmental Toxicology, University of California at Davis, March1, 1989.

SELECTED BIBLIOGRAPHY (about 200 publications):

Recent Examples

1. Malins, DC, Holmes, EH, Polissar, NL, Gunselman, SG. The etiology of breast cancer: Characteristic alterations in hydroxyl radical-induced DNA base lesions during oncogenesis with a potential for evaluating incidence risk. Submitted for publication, 1992.

2. Malins, DC. Identification of hydroxyl radical-induced lesions in DNA base structure: Biomarkers with a putative link to cancer development. Submitted for publication, 1992.

3. Malins, DC, Haimanot R. Major alterations in the nucleotide structure of DNA in cancer of the female breast. 1991; 51: 5430-5432.

4. Malins, DC, Haimanot R. The etiology of cancer: Hydroxyl radical-induced DNA lesions in histologically normal livers of fish from a population with liver tumors. 1991; Aquatic Toxicol; 20: 123-130.

5. Malins DC, Ostrander GK. Perspectives in Aquatic Toxicology. Annual Reviews in Pharmacol and Toxicol 1991; 31:371-399.

6. Malins DC, Haimanot R. 4,6-Diamino-5-formamidopyrimidine, 8-hydroxyguanine, and 8-hydroxyadenine in DNA from neoplastic liver of English sole exposed to carcinogens. Biochem Biophys Res Commun 1990; 173:614-619.

7. Malins DC, Ostrander GK, Haimanot R, Williams P. A novel DNA lesion in neoplastic livers of feral fish: 2,6-diamino-4-hydroxy-5-formamidopyrimidine. Carcinogenesis 1990; 11: 145-147.

8. Malins DC, McCain BB, Brown DW, Chan S-L. Toxic chemicals in marine environments: Food-chain transfers and biological effects. In: Health and environmental research on complex organic mixtures (Proc 24th Hanford Life Sci Symp) 1987; 71: 5-16.

9. Malins DC, McCain BB, Myers MS, Brown EW, Krahn MM, Roubal WT, Schiewe ML, Landahl JT, Chan S-L. Field and laboratory studies of the etiology of liver neoplasms in marine fish from Puget Sound. Environ Health Perspectives, 1987; 71: 5-16.

10. Malins DC, McCain BB, Brown DW, Myers MS, Krahn MM, Chan S-L. Toxic chemicals, including aromatic and chlorinated hydrocarbons and their derivatives, and liver lesions in white croaker (Genyonemus lineatus) from the vicinity of Los Angeles. Environ Sci Tech 1987; 21(8): 765-770.

11. Malins DC, Krahn MM, Brown DW, Rhodes LD, Myers MS, McCain BB, Chan S-L. Toxic chemicals in marine sediment and biota from Mukilteo, Washington: Relationships with hepatic neoplasms and other hepatic lesions in English sole (Parophyrs vetulus). J Nat Cancer Inst 1985; 74(2): 487-494.

Exhibit E

BEFORE THE
UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
REGION I

In Re:	)	No. _____
LINCOLN PULP & PAPER COMPANY NATIONAL POLLUTANT DISCHARGE ELIMINATION SYSTEM Permit No. ME0002003	)	

DECLARATION OF DR. RICHARD W. CLAPP

1. My name is Richard W. Clapp. I am a resident of Boston, Massachusetts. Since 1989, I have been Director of the JSI Center for Environmental Health Studies. The JSI Center is part of JSI Research and Training Institute, a non-profit public health consulting firm located in Boston, Massachusetts. I am also Associate Professor in the Department of Environmental Health and teach courses at the Boston University School of Public Health.

BACKGROUND AND CREDENTIALS

2. My formal education and training can be summarized as follows. I have a doctorate in epidemiology from the Boston University School of Public Health. I received a Masters Degree in Public Health from the Harvard School of Public Health. My undergraduate degree from Dartmouth College was in Biology.

3. I have published a number of articles in professional, peer-reviewed journals. Approximately fifteen publications focus on the causes of cancer or studies of the patterns and distributions of cancer in different populations. Several of these articles focus on cancers in Vietnam veterans who have been exposed to the herbicide "Agent Orange."

4. The primary contaminant of Agent Orange is dioxin. I have studied the scientific literature concerning the effects of dioxin on animals and humans. In addition, I have attended international conferences on the toxic effects of dioxin and dioxin-like chemicals. At the request of the Veterans Administration and the Centers for Disease Control, I have reviewed documents that discussed the health effects of dioxin.

5. I have conducted studies of cancer in people exposed to dioxin and dioxin-like compounds. Similarly, one of my colleagues at Boston University has studied the reproductive effects of dioxin on children whose parents were exposed to dioxin on the job or as military personnel in Vietnam. Other studies I have participated in have examined neurological, immunological, enzyme, and endocrine effects of dioxin and related chemicals.

6. At the request of the U.S. Environmental Protection Agency (EPA), I participated in the agency's effort to reassess the health effects of dioxin (1994 Draft Dioxin Reassessment). This work involved reviewing scientific evidence regarding the cancer and non-cancer health effects of dioxins and related compounds. The majority of the work was concluded in the Fall of 1995.

NON-CANCER EFFECTS OF DIOXIN

7. The nature of dioxin's adverse effects, including the full range of non-cancer effects on the immune, reproductive/hormonal and neurological systems, is much better understood as a result of research in the past few years. It is now widely believed by scientists involved in dioxin research that non-cancer effects may occur at exposure levels even lower than have been assumed to result in "acceptable" risk of cancer. Furthermore, for several of these non-cancer effects caused by dioxin, there may not be a threshold level. This means that any level of exposure above zero may trigger an adverse impact in the body.

8. Current levels of dioxin in the average U.S. human body resulting from on-going daily exposures may already be high enough, without additional dioxin discharges, to trigger non-cancer health effects in humans. EPA has concluded in the 1994 Draft Dioxin Reassessment that current average dioxin exposures nationally are 1-2 orders of magnitude higher than any reference dose (virtually safe dose) that might be calculated for dioxin. More highly exposed populations, like members of the Penobscot Indian Nation who have fished for subsistence in waters heavily contaminated by dioxin, would have even higher body burdens than the national average.

9. The EPA, in the 1994 Draft Dioxin Reassessment, concludes that a reference dose, if it were to be set, would be less than 1 picogram (trillionth of a gram) of dioxin per kilogram of body weight per day. The Federal Agency for Toxic Substances and Disease Registry (ATSDR) established a minimal risk level for adverse effects of 1 pg/kg/day in its Toxicological Profile for Dioxin published in 1989. The ATSDR also notes that the most vulnerable populations may be the fetus and breast-fed babies, because of the developmental and reproductive toxicity of dioxin. The EPA is expected to finalize its Draft Dioxin Reassessment in the coming year. Prior to this, public health or environmental agencies should not use a reference dose for dioxin less protective than the ATSDR 1 pg/kg/day value.

10. Given these circumstances, the complete omission by EPA of any analysis of dioxin's non-cancer adverse health effects is

disturbing. The further complete omission of any consideration of the effects of other dioxin-like compounds, other routes of exposures other than the water column and fish consumption, and sensitive subpopulations, such as developing fetuses and nursing infants, compounds the problem for public health decision-makers. As a result, the current risk assessment for the Lincoln mill permit is useless as a basis for decisions by public health professionals and agencies that must determine the acceptability of risks posed by the permit.

11. In my opinion, as a public health professional, there is no safe level for dioxin exposure. The prudent position that most public health professionals take with regard to carcinogens like dioxin is to strive to limit exposure to zero. Current levels of dioxin in the bodies of U.S. citizens as a result of on-going daily exposures are already high. It is not justified, from a public health point of view, to permit the operation of additional facilities that will discharge more dioxin. The only way to reduce current cancer rates is to reduce carcinogenic exposures wherever possible.

12. In my opinion, incomplete risk assessments cannot be used to determine whether a particular population's health will be protected from an action. Risk assessments were initially designed to assist decisionmakers in ranking priorities. Properly conducted risk assessments that look at all risks may be helpful for this purpose. In the case of dioxin, background levels and body burdens are at the level or within an order of magnitude of the level where health effects occur.

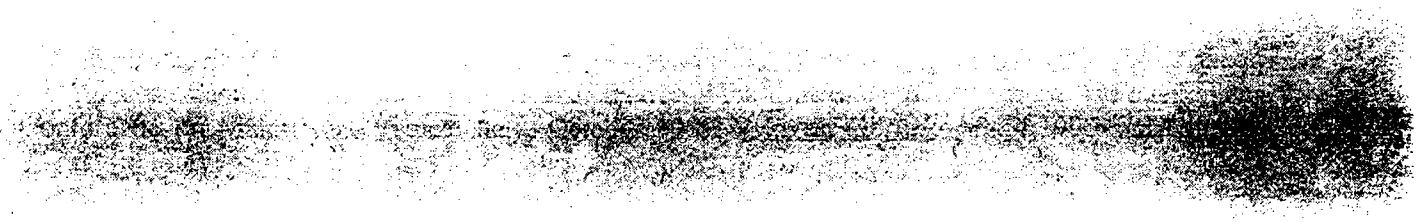
13. The decision to expose people to additional dioxin discharges presents a fundamental policy choice. In my opinion, dioxin levels are already too high and the health effects too severe to allow further exposures, particularly where alternatives to the action exist. Health policymakers made the decision not to allow additional exposures to lead for precisely this reason. This fundamental policy decision should not be masked by a risk assessment theorectical exercise that looks at only some of the known impacts and then seeks to justify what would, under any credible public health inquiry, be considered as serious health risks.

Pursuant to 28 U.S.C. § 1746, I, Dr. Richard W. Clapp, hereby declare under penalty of perjury, that the foregoing is true and correct.

Executed on February 27, 1997.

Richard W. Clapp

Richard W. Clapp, MPH, D.Sc.



RICHARD W. CLAPP

EDUCATIONAL EXPERIENCE

Sc.D	Boston University (1989) School of Public Health Epidemiology
M.P.H.	Harvard School of Public Health (1974) Health Services Concentration
A.B.	Dartmouth College (1967) Biology Concentration

PROFESSIONAL EXPERIENCE

1992 - present

Boston Univ. School of Public Health
Dept. of Environmental Health
80 E. Concord St.
Boston, MA

Associate Professor. Teach courses in environmental health and environmental epidemiology to masters and doctoral level graduate students. Advise doctoral students on dissertations in environmental health and epidemiology. Participate in departmental committees and research activities, including assessment of health effects of nuclear weapons production, lead paint removal and electromagnetic radiation.

1994 - present

John Snow, Inc.
210 Lincoln Street
Boston, MA

Consultant. Provide expert advice and training programs for citizens groups and interested professionals on health effects of environmental toxic exposures in communities. Participate in planning new initiatives and in conducting international environmental health consulting and training activities.

1989 - 1994

John Snow, Inc.
210 Lincoln Street
Boston, MA

Director of Center for Environmental Health Studies. Responsible for development and conduct of studies of health effects of environmental toxic exposures in communities. Coordinated consultants from Boston University School of Public Health Environmental Health Department providing expert advice and training programs for citizens groups and interested professionals. Managed personnel and budget for variety of projects.

1980 - 1989 Massachusetts Department of Public Health
Massachusetts Cancer Registry

Director. Responsible for establishing statewide cancer incidence reporting system, coordinating reports from over one hundred fifteen hospitals and licensed clinics, and centralizing information in computerized database. Supervised staff and consultants involved in data editing, quality assurance and data reporting activities. Worked with broad-based advisory committees, citizens groups, and epidemiologic researchers conducting studies of cancer incidence in Massachusetts. Involved in numerous Department of Public Health committees and research projects, including leukemia in Woburn, and other cities and towns. Participated in regional and national organizations of cancer registry directors.

1979 - 1980 Equifax Health Systems Division
Reading, MA

Acting Director of Occupational and Environmental Health Studies. Participated in epidemiologic feasibility study of health effects of low-level ionizing radiation, review of OSHA health standards for lead, cotton dust, and asbestos, review of comments on Federal inter-agency carcinogens policy. Supervised staff involved in evaluating union-based occupational health education grant and surveying U.S. population-based cancer registries.

1977 - 1978 Massachusetts Department of Public Health
Childhood Lead Poisoning Prevention

Director. Supervised laboratory, office, field inspector and legal staff of statewide program involved in screening for lead poisoning and investigating possible environmental sources of lead. Coordinated development of job training programs for unemployed persons in the areas of lead paint

inspections and lead hazard abatement in dwellings. Reported to Governor's Committee on Childhood Lead Poisoning and managed diverse personnel and budgets. Presented educational programs and videotaped training sessions on childhood lead poisoning.

1975 - 1976 Lynn Community Health and Counseling Center
Lynn, MA

Executive Director. Responsible for overall management of multi-service center offering comprehensive pediatric and adolescent health services, family planning services, childhood lead poisoning prevention services, individual and family counseling, social service advocacy and a day activity program for mentally retarded adults. Worked with other human services agencies in developing a WIC program, and participated in regional and state-level health planning activities. Reported to community board and managed diverse personnel and budgets.

1974 - 1975 Massachusetts General Hospital
Boston, MA

Manager, Pediatric and Psychiatric Group Practices. Managed conversion of out-patient clinics to hospital-based group practices with salaried staff as part of developing Ambulatory Care Center. Implemented cost centers and program planning and budgeting system and reported to Medical Directors of two specialty groups.

1972 - 1974 Massachusetts Department of Public Health

Deputy Director, Prison Health Project. Hired medical and ancillary health staff for five state prisons, supervised survey of prison health conditions in county and municipal correctional facilities, and coordinated establishment of two community-based alternative programs for inmates convicted of drug-related crimes. Established twenty-four hour emergency coverage for maximum security prison, and worked with inmate medical advisory committees at several facilities.

1970 - 1972 New York City Health Services Administration

Program Research Analyst. Analyzed public health programs in City Hospitals, the prison hospital and Houses of Correction. Made recommendations regarding improved operations and staffing levels. Drafted guidelines for affiliation agreement for teaching hospital administration of Riker's Island prison medical services.

TEACHING APPOINTMENTS

Tufts University School of Medicine - Assistant Clinical Professor, 1989-present.

Boston University School of Public Health - Adjunct Assistant Professor, 1990-1993; Assistant Professor, 1993-1995, Associate Professor, 1995-present.

PROFESSIONAL MEMBERSHIPS

American Public Health Association, Society for Epidemiologic Research, Massachusetts Public Health Association, Harriet Hardy Institute, MassCOSH, International Society for Environmental Epidemiology, American College of Epidemiology.

PUBLICATIONS

Clapp, RW. "The Massachusetts Childhood Lead Poisoning Prevention Program," in Low Level Lead Exposure: The Clinical Implications of Current Research, Needleman HL, ed., Raven Press, NY, NY, 1980.

Dreyer NA, Loughlin JE, Friedlander ER, Clapp RW, Fahey FH. "Choosing Populations to Study the Health Effects of Low-Dose Ionizing Radiation," Am J Pub Hlth 71: 1247-1252, 1981.

Finison L, Jaques P, Spaight S, Fine W, Clapp RW, O'Sullivan V. "Data Bases for Patterns of Care Studies in Defined Populations." Prog Clin Biol Res 130:465-476, 1983.

Kogan MD, Clapp RW. "Soft Tissue Sarcoma Mortality Among Vietnam Veterans in Massachusetts, 1972 to 1983." Int J Epidemiol 17: 39-43, 1988.

Hesketh PJ, Clapp RW, Doos WG, Spechler SJ. "Increasing Frequency of Adenocarcinoma of the Esophagus," Cancer 64:526-530, 1989.

Longnecker MP, Clapp RW, Sheahan K. "Associations Between Smoking Status and Stage at Diagnosis of Colo-rectal Cancer, Massachusetts, 1982-1987," Cancer 64: 1372 - 1374, 1989.

Koh HK, Clapp RW, Prout MN, et al. "Systematic Underreporting of Cutaneous Malignant Melanoma in Massachusetts: Possible Implications for National Incidence Figures." J Am Acad Dermatol 24(4): 545-550, 1991.

Clapp RW, Cupples LA, Colton T, Ozonoff DM. "Cancer Surveillance of Veterans in Massachusetts, USA, 1982 - 1988." Int J Epidemiol 20: 7-12, 1991.

Koh H, Clapp R, Barnett J, Prout M, Geller A, and Lew R: "Systematic underreporting of cutaneous malignant melanoma: Implications for incidence figures in the United States." Abstract, 2nd International Conference on Melanoma, Venice, Italy, October 1989; J Invest Dermatol 94;4:1990 and Clin Res 38:659A, 1990 (Abstract).

Koh H, Geller A, Miller D, Clapp R, Mercer MB, Lew R: "Who discovers melanoma? Patterns from a population-based survey." J Am Acad Dermatol 26:914-9, 1992.

Clapp RW, and Olson JR: "A New Review of the Dioxin Literature in the Context of Compensation for Vietnam Veterans." New Solutions 1;4: 31-37, 1991.

Geller A, Koh H, Miller D, Clapp R, Mercer M, and Lew R: "Use of Health Services before the Diagnosis of Melanoma: Implications for Early Detection and Screening." J Gen Intern Med 7:154-7, 1992.

Swartz J, and Clapp R: "New Cancer Theories: Policy Implications for Cancer Prevention." New Solutions 2;4:17-21, 1992.

Longnecker MP, Newcomb PA, Mittendorf R, Greenberg ER, Clapp RW, et al: "The Reliability of Self-reported Alcohol Consumption in the Remote Past." Epidemiology 6:535-539, 1993.

McConnell R, Anderson D, Russell W, Anderson K, Clapp R, et al: "Angiosarcoma, porphyria cutanea tarda and probable chloracne in a worker exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin-contaminated waste oil." Br J Ind Med 50:699-703, 1993.

Newcomb P, Longnecker MP, Mittendorf R, Greenberg ER, Clapp RW, et al. "Lactation and a Reduced Risk of Premenopausal Breast Cancer." New Eng J Med 330(2): 81-87, 1994.

Mittendorf R, Longnecker MP, Newcomb PA, Dietz AT, Greenberg ER, Bodgan GF, Clapp RW, Willett WC. Strenuous Physical Activity in Young Adulthood and Risk of Breast Cancer. Cancer Causes Control 6: 347-353, 1995.

Coogan P, Clapp R, Wenzl T, Newcomb P, Longnecker M. Occupational Exposure to 60-Hz Magnetic Fields and Risk of Breast Cancer in Women. Am J Epidemiol 141(11):S32 (abstract), 1995.

Longnecker MP, Newcomb PA, Mittendorf R, Greenberg ER, Clapp RW, Bogdan GF, Baron J, MacMahon B, Willett WC. Risk of Breast Cancer in Relation to Lifetime Alcohol Consumption. J Natl Cancer Inst 87(12): 923-929, June 21, 1995.

Proctor S, Clapp R, Coogan P. Prevalence of Depressive Symptoms in a Survey of Aluminum Workers. New Solutions 5(4); Summer, 1995.

Newcomb PA, Longnecker MP, Storer BE, Mittendorf R, Greenberg ER, Clapp RW, Bogdan GF, Willett WC. Long-term Hormone Replacement Use and Risk of Breast Cancer in Postmenopausal Women. Am J Epidemiol 142:788-795, 1995.

Geller A, Miller D, Lew R, Clapp R, Wenneker M, Koh H. Cutaneous Melanoma Mortality among the Socioeconomically Disadvantaged in Massachusetts. Am J Public Health 86:538-543, 1996.

Baron JA, Newcomb PA, Longnecker MP, Mittendorf R, Storer BE, Clapp RW, Bogdan G, Yuen J. Cigarette Smoking and Breast Cancer. Cancer Epi Biomarkers Prev (in press).

Coogan PF, Clapp RW, Newcomb PA, Wenzl TB, Bogdan G, Mittendorf R, Baron JA, Longnecker MP. Occupational Exposure to 60 Hz Magnetic Fields and Risk of Breast Cancer in Women. Epidemiology 7:459-464, 1996.

Coogan PF, Clapp RW, Newcomb PA, Mittendorf R, Bodgan G, Baron JA, Longnecker MP. Variation in Female Breast Cancer Risk by Occupation. Am J Ind Med 30:430-437, 1996.

Newcomb PA, Longnecker MP, Storer BE, Mittendorf R, Baron J, Clapp RW, Trentham-Dietz A, Willett WC. Recent Oral Contraceptive Use and Breast Cancer (United States). Cancer Causes and Control 7:525-532, 1996.

PRESENTATIONS

"Cancer Surveillance in Massachusetts, 1982-1983." International Association of Cancer Registries Meeting, Hartford, CT, 1985.

"Dealing with Cancer Clusters." American Association of Central Cancer Registries founding meeting, Chicago, IL, 1988.

"Statistical Methods for Analyzing Cancer Clusters." National Conference on Clustering of Health Events, Atlanta, GA, 1989.

"Cancer Statistics and the Right to Know". American Public Health Association Annual Meeting, Boston, 1988.

"Respiratory Disease Mortality and Morbidity, Respiratory Cancer and Mesothelioma Incidence: Occupational Associations in Massachusetts, 1982-1985." American Public Health Association Annual Meeting, Boston, 1988.

"Soft Tissue Sarcoma Incidence in Massachusetts Vietnam Veterans, 1982-1986." American Public Health Association Annual Meeting, Boston, 1988.

"Respiratory Cancer by Race and Gender: Selected Occupational Associations in Massachusetts, 1982-85,"

National Minority Health Conference, Atlanta, 1990.

"Occupation and Race Data in Central Cancer Registries," American Public Health Association Annual Meeting, Atlanta, 1991.

"New Carcinogen Threshold Theories: Implications for Prevention," University of Connecticut conference on Incorporating Molecular Mechanisms into Estimates of Cancer Risk, 1992.

"Angiosarcoma, porphyria cutanea tarda and probable chloracne in a worker exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin-contaminated waste oil," Twelfth International Symposium on Dioxins and Related Compounds, Tampere, Finland, 1992.

"Popular Epidemiology." Loka Institute Conference on Dissenting Ways of Knowing, University of Massachusetts, Amherst, MA, 1994.

"Agency Responses to the Woburn leukemia cluster". Fifth Annual Symposium on Environmental and Occupational Health during Societal Transition in Central and Eastern Europe, Nitra, Slovak Republic, 1994.

"The Upper Cape Cancer Incidence Study". Sixth Annual Symposium on Environmental and Occupational Health during Societal Transition in Central and Eastern Europe, Eforie Nord, Romania, June, 1995.

OTHER INVITED PAPERS

"Cancer Surveillance of Vietnam Veterans in Massachusetts". Distinguished Lecture Series in Occupational Medicine. Robert Wood Johnson Medical School, Piscataway, NJ, 1989.

"Patterns of Cancer in Vietnam Veterans". Hematology/Oncology Rounds. Massachusetts General Hospital, Boston, MA, 1991.

"Agent Orange, Health Effects and Government Policy". Health and the Environment Lectureship. Brown University, Providence, RI, 1991.

"Agent Orange and Cancer". Cancer Prevention Rounds. Boston University Medical Center, Boston, MA, 1994.

"Agent Orange and Veterans Health - 1996 Update." Public Health Forum, Boston University School of Public Health, Boston, November, 1996.

HONORS AND AWARDS

Lemuel Shattuck Award, Massachusetts Public Health Association, 1990
Environmental Health Network 1990 National Award
Member of Massachusetts Advisory Board on Toxics Use Reduction, 1990-1994
Chair, Massachusetts Toxics Use Reduction Institute Science Advisory Board, 1994-1996